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DEPARTMENT OF THE ARMY
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CONTRIBUTIONS TO THE THEORY OF ACID POISONING

[Following is a translation of selected portions of an article by Docent Dr. R. von Limbeck, Imperial and Royal Chief Physician, of the Fourth Medical Division of the Imperial and Royal Rudolf Foundation Hospital in Vienna, in the German-language periodical Zeitschrift für Klinische Medicin (Journal of Clinical Medicine), Vol 34, No 5-6, pages 419-468.]

As is well known, the modern theory of the toxic effect in the body of acids ingested into it is based, apart from earlier works (Salkowski, Gaethgens), principally on the investigations carried out by Walter, a pupil of Schmiedeberg's, on this subject. [Archiv für Experimentelle Pathologie und Pharmakologie (Archives of Experimental Pathology and Pharmacology), Vol 7.] According to Walter, organisms behave differently toward acids that are introduced into the stomach and cannot be oxidized depending on whether they belong to the class of herbivorous or carnivorous animals. Introduction of acid causes in the rabbit a decrease in the carbonic acid content of the blood, the fixed alkali of the blood combining with the acid administered and the carbonic acid formed in the tissues no longer encountering the sufficient quantity of fixed alkalies with which to circulate in combination. That, according to Walter, is the way the toxic effect of acid comes about in the rabbit. It is otherwise with dogs. The relative richness of their bodies in ammoniac protects the alkalies of the blood, and the acid introduced leaves the body, largely in combination with the afore-mentioned ammoniac. No combination of blood alkalies with the acids introduced comes about in these animals, he says; the carbonic acid in the blood of dogs is very little diminished after administration of acid. Later studies [Coranda, Archiv für Exper. Pathologie und Pharmakologie, Vol 12; Hallervorden, ibid., Vol 10 and 12] in general taught with reference to man, too, a reaction analogous to that of the canine

organism. Not only did excessive acid in the diet of man, too, lead to an increase in the elimination of ammoniac in the urine, but such an increase was also found in certain intoxications and febrile diseases as a very common though not ever-present phenomenon whose cause has so far been taken to be an increased acidification of the tissues. The ammoniac of the urine thus actually plays the part of acid indicator of the tissues in the dog and in man (Hallervorden).

While these observations appeared to set forth the undoubtedly high significance of ammoniac in the neutralization of acids formed in the body or introduced into it, nevertheless the conditions prevailing were still not yet adequately clarified. Walter himself found that in his dogs only a part of the acid administered was covered by the extra ammoniac, and gave it as his opinion that the rest of it served in part to increase the acidity of the urine and in part caused a slight removal of fixed alkalies from the blood. Coranda observed the same thing in an experiment on human subjects, and he expresses the conjecture concerning the remaining acid not covered by the ammoniac that either it had not been resorbed or the increased elimination of ammoniac lasted longer than it was measured. Apart from the abode and the effects of this remaining acid on the metabolism of the body, nothing was known of influences in other directions of acid introduced into human and animal bodies. This gap was filled for the dog later by two experiments conducted by Gaethgens [Zeitschrift für Phys. Chemie (Journal of Physical Chemistry), Vol 4). For this side of human metabolism we have only two very recent articles by Dunlop [Journal of Physiology, Vol 14] and Biernacki [Münchener Medicinische Wochenschrift (Munich Medical Weekly), 1896].

Gaethgens reports the following experiments:

Two experiments were performed on a dog weighing 20 kg, its food in the first consisting of leached-out and in the second of fresh horsemeat. In the first experiment on the 9th day and in the second on the 8th, 9th, and 10th the dog was given sulfuric acid, the amount given being 7 g in the first experiment and a total of 16.0 in the second of sulfuric acid hydrate. In both cases the acids and bases in the animal's urine were measured daily side by side. The experiments yielded the following results: Introduction of acid into the organism of the dog effects an increase in the ammoniac content of the urine. The additional quantity of this substance eliminated in the first experiment was sufficient to cover the requirements of the acids conveyed into the urine, insofar as they were not taken up by small quantities of fixed bases, for the formation of ammonium salts. In the second experiment, in which the dog had been fed fresh horsemeat for 8 days before and during the experiment, i.e. had had an acid diet, in order to set the ammoniac content of its tissues as low as possible, great quantities of fixed bases were removed from the organism in the urine by the administration of acid.

These experiments show that in dogs the ammoniac takes care of the neutralization of experimentally introduced acids exclusively or almost exclusively only under certain conditions, but that otherwise, when the body does not have sufficient quantities of this substance available, fixed bases also leave the body in combination with the acid introduced, just as in the case of herbivorous animals.

The circumstance that man, as is generally assumed, is much closer to the dog with regard to metabolism than to herbivorous animals, would make it probable that his reaction to the introduction of acid might be more like that of the dog than that of a herbivorous animal. The above-mentioned conditions of ammoniac elimination in man after the introduction of acid told in favor of this view. But as to whether the introduction of acid also occasions losses of fixed alkalies in man, to the best of my knowledge no information existed in the literature until very recently. Only in the last few years has one brief report each been made by Dunlop and Biernacki which deal with this subject and in which both authors indicate an increase in elimination of fixed alkalies in tho urine as a result of administration of acids per os in man. The importance of such a finding for the theoretical conception of the effects of acid in man moved me to check the existing indications in this direction. But since the above-mentioned authors who experimented on man, Dunlop and Biernacki, have composed their reports up to now in very brief form and apart from the fixed alkalies have taken account of few (Dunlop) or no (Biernacki) other substances in the urine, it seemed to me desirable to undertake experiments on man after the example of Gaethgens in such a way that before and after the introduction of the acid an accurate understanding would be gained of the constitution not only of the urine but if possible also of the faeces.

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B. Clinical Observations

Beside the relative meagerness of the literature on the consequences of experimental administration of acids, the great abundance of clinical reports on the existence of acid poisoning in human patients cannot escape notice. When we look for the foundations of such opinions, it turns out that aside from isolated findings of abnormal acids in the urine of the patients in question (hydroxybutyric acid, sarcolactic acid, hydroxymandelic acid, etc.), in the majority of cases the conclusion as to the occurrence of acid poisoning was only drawn on the basis of other, in general not always reliable evidence. This is especially true of the observations of alleged reduced alkalescence of the blood by which it has been attempted to support the acid theory of various pathological processes. If for the

moment with respect to determination of the alkalescence of the blood we disregard gas analysis methods, the most recent literature [Löwy, Pflüger's Archiv, Vol 58; Limbeck, Wiener Medicinische Blätter (Vienna Medical Papers)] has shown that the alkalimetric titration methods of Landois, von Jaksch, Kraus, and others, by which until recently observations of the occurrence of diminished alkalescence of the blood were almost always made in connection with such processes, give incorrect results. Löwy [Centralblatt für Medicinische Wissenschaften (Central Journal of Medical Sciences), 1894, page 785] and also Limbeck and Steindler [Centralblatt für Innere Medicin (Central Journal of Internal Medicine), 1895, No 27] among others have shown that with more correct methods of alkalimetry of the blood and the serum those pathological processes in which heretofore the reduced alkalescence of the blood was held as a dogma do not in fact regularly show such reduced alkalescence either in the serum or in the whole blood. It is another matter with the second method, usually used clinically for judging as to the occurrence of acid poisoning, namely determination of the ammoniac in the urine. Although the methodological and theoretical bases of this method are far superior in reliability to the alkalimetry of the blood as practiced until quite recently, still experimental and clinical observations have shown that ammoniac by its increase in the urine indicates often, but not reliably, increased acidification of the tissues, since in febrile processes there are periods within which very probably abnormal acids or an excess of normal acids may circulate in the body without an increase in the ammoniac in the urine either absolutely or in relation to the total nitrogen. Such cases are to be found both among those compiled by Hallervorden and among Gumlich's [Zeitschrift für Phys. Chemie, Vol 17, page 10] and recently again in Rumpf's [Virchow's Archiv, Vol 143, page 1]. This seems to be observed with quite special frequency in croupous pneumonia, the ammoniac figure in the urine often remaining normal before the crisis, and NH_3 increase only setting in in the urine after the crisis. We can see from these observations that the amount of ammoniac in the urine, at least at certain periods and in certain infectious diseases, is not an absolutely sure measure for the occurrence of over-acidification of the tissues of the body, either. We do not yet know whether all ammoniac compounds of the acids pathologically formed in the body leave the body equally quickly in the urine. Only in that case could a rise in the amount of ammoniac in the urine actually be counted as the surest sign of increased acidification of the tissues.

Under such circumstances it seemed to me desirable on the basis of results obtained concerning the influence of experimental administration of acid on the metabolism of the healthy human subject to compare these findings with observations

of sick persons, especially "acid poisoned" ones, and look for analogy between the changes to be observed. A rise in elimination of nitrogen and fixed alkalies in the urine above the intake would be the most important signs apart from a rise in ammoniuria of deviation of the acid-conditioned human metabolism from the norm. There was also a second circumstance that moved me to undertake alkali determinations in the urine of the sick.

That was the almost complete lack today of knowledge of the quantities of fixed alkalies eliminated by sick persons. It is true that Salkowski's measurements [Virchow's Archiv, Vol 53], carried out with great accuracy and enormous industry, are available in the literature, where the author investigated the quantities eliminated and the relative proportion of the K and Na bases in the urine of fever patients, but today if we wished to make use of these figures to answer the question set above as to the alkali balance of the body, we should miss any measurement of intake. Salkowski did not at that time report the alkali content of the diet or even the diet itself, so that his figures are impossible to use for an alkali balance. He found that in general the quantity of alkalies in the urine of fever patients was diminished. The potassium was usually less affected by this reduction during the first period of the illness than the sodium, which he often found to decline to a minimum during the period of fever. How far this decrease in fixed alkalies in the urine was dependent on the patient's simultaneous condition of hunger is, as has been mentioned, not to be seen from Salkowski's observations.

The twelve cases described below were with one exception investigated from these standpoints. The reason for the exception will be discussed later. For the sake of clarity I have divided the cases into three groups: 1) poisonings, 2) febrile processes, and 3) neoplasms.

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II. Infectious Diseases Accompanied by High Fever

Since the appearance of the gas analysis studies of Senator [Untersuchungen über den Fieberhaften Process (Studies Concerning the Febrile Process), Berlin, 1873], Geppert [Zeitschrift für Klinische Medicin (Journal of Clinical Medicine), Vol 2, page 364], Minkowski [Archiv für Experimentelle Pathologie und Pharmakologie, Vol 19, page 233], Kraus [Zeitschrift für Heilkunde (Journal of Therapeutics), Vol 10], and Klemperer [Charité-Annalen (Charité Annals, i.e. annals of the Charité hospital in Berlin), Vol 15, page 151] and the long series of publications which have dealt with alkalimetric measurements of the blood of human patients, the proposition that fever in man as in the dog is accompanied by a decrease in CO_2, or a decrease in the alkalescence, i.e. the capacity of the blood

to combine with acids, has been accepted as dogma.

More recently doubts have been raised about such an assumption by Löwy, Steindler, and me, and later by Strauss [Zeitschrift für Klinische Medicin, Vol 30, page 317], Berend [Zeitschrift für Heilkunde, Vol 17, page 351] and others on the basis of new titrimetric measurements of the blood, and Biernacki [Zeitschrift für Klinische Medicin, Vol 31, page 1] has found that in the method of examining defibrinated blood usual up to now not all the CO_2 of human blood is found by the gas pump. Defibrinated blood is always poorer in CO_2 than uncoagulated oxalated blood. The decline in CO_2 in fever can be demonstrated only in defibrinated blood, not in uncoagulated blood. If the question of a regular link between feverish body temperature and signs of over-acidification of the tissues thus cannot yet be answered with certainty today by examination of the blood, the next thing seems to be to pursue the same goal by other means.

As far as investigation of the metabolism of fever patients is concerned, that has already been done, and since Hallervorden's studies the heightened ammoniuria in particular has been taken as a sign of the occurrence of acidification. It was pointed out at the beginning of this section that NH_3 determination is not an infallible method of testing this question. It was therefore the purpose of the investigations reported below to find new criteria in the balance of the total nitrogen, the quantitative variation of the N (NH_3), the balance of the alkalies (at least for the urine) for whether every patient that shows fever temperatures shows signs in his urine of over-acidification of his tissues or not.

From this point of view six patients with high fevers, the histories of whose illnesses are given below, were observed by me as closely as possible, in most cases for 5 days insofar as was necessary and possible. The results of these studies, which thus deal with the relationships between feverish rises in temperature and the occurrence of symptoms of abnormal acid formation in the body, I should like to summarize here briefly in advance. It was found that there are sick persons with high fevers who during the period of fever do display in the urine the typical picture of over-acidification of the tissues. It seems to me no less firmly established, however, that patients with equally high fever showed not the slightest signs of increased acid effects throughout the 5-day duration of the study, despite the fact that a high body temperature prevailed the whole period. Besides this, however, still other types of peculiarities appear to exist, either in different infectious diseases or perhaps in different individuals, for signs of increased acid elimination in the urine set in in one case during the period of fever, and in another case only after the fever had

passed. In still a third direction, too, there are differences, in that ammoniac and alkalies are not always equally involved, in cases of patients with fever where signs of acid poisoning do occur, in the increased elimination in the urine; sometimes it is chiefly the ammonium salts that are concerned, but sometimes the alkalies are more strongly affected. What the basis of this last phenomenon is cannot yet be said with certainty. The assumption strongly suggests itself, however, that the different acids pathologically formed in the body can by combining with the bases form the most varied salts, whose diuretic threshold, whose toxicity, etc., may vary widely. Along this line we already have the observations of Marfori [Archiv für Experimentelle Pathologie und Pharmakologie, Vol 33], who found that various ammonium salts are converted by dogs and rabbits in quite different ways into uric acid and are also tolerated in varying degrees.

1. A Case of Febris Intermittens Tertiana

K. Franz, 18-year-old handyman, admitted 12 June, discharged as cured 21 June. According to his statement the patient had always been in good health except for a case of scarlet fever he suffered in early childhood. Three days before he had become ill without any cause known to him, with a trembling chill, high fever, and an outbreak of sweat; the next day he felt very weak but had no fever. On the day of his admission a chill and fever again set in early in the afternoon; this persuaded him to come to the hospital.

Upon admission he was found to have a high fever (39.6° C). The organs of the thorax showed no further change, the abdomen was not swollen, but the spleen was palpable and from percussory indications definitely enlarged. The liver showed no peculiarities and the urine no pathological constituents. The blood, repeatedly examined in both a fresh and a fixed state, always showed the presence of malaria parasites of the tertian form.

Because of external circumstances, although the patient again was sick with a chill punctually on 14 June, his urine did not come under examination with a regulated diet until 16 June, on which day a new attack was in prospect for the afternoon. On the next fever-free day, 17 June, the examination was repeated, but then on 18 June the patient was given 0.5 g of quinine, and from then on, with continued administration of this preparation, had no more chills and remained free of fever until his discharge.

The study of this case thus covers only two days, one a fever day and one fever-free. Short as the time was, the result seemed to me to be quite clear. Another remarkable thing about this case is that in spite of the fever the patient had a good appetite even on 16 June and took the same quantity and quality of food as on the next fever-free day [see note].

[Note] The food given the patient on each of the two days of observation and eaten by him both times consisted daily of: milk 200 ccm, bouillon 300 ccm,

ham (lean) 100 g, white bread (without crust) 120 g, egg 100 g, and veal (roast) 100 g.

The table below shows the results of examination of the urine for the two days indicated.

Table VIII

July	Amount of Urine	Density	N	$N(NH_3)$		KCl + NaCl	K_2O	Na_2O	CaO	HCl	P_2O_5	SO_3	Temperature	
				g	% of Tot. N								a.m.	p.m.
16	2250	1008	14.611	1.114	7.62	14.259	2.937	5.196	0.119	7.714	1.083	0.538	36.2	37.2*
17	2570	1007	13.31	0.899	6.75	5.643	0.717	2.406	0.282	6.992	2.762	0.580	36.3	36.4

*Chill at 1:45 p.m. Temperature at 3:00 p.m. 40.5° C.

When one looks at this little table the differences in the eliminations in the urine in the second and third columns are hardly noticeable. In the composition of their components, however, the two are clearly and -- as will be observed -- characteristically different. This is especially true of the alkalies, the chlorides and phosphates.

The total elimination of nitrogen is a little greater on the fever day than on the fever-free one, but on both days the elimination of N in the urine exceeds the amount of N introduced in the food served, though by very little (daily intake of N ca. 12.0 g N). The ammoniac is somewhat high on both days as compared to the total nitrogen present, though not very. The greatest difference is shown by the alkalies KCl + NaCl, which were taken in on both days in the food in the amount of about 10 g, so that on the fever day a loss of over 4 g took place through the urine, while on the fever-free day as far as the urine is concerned a retention of alkalies in the body could have taken place. Whether this actually occurred and on what scale I am not in a position to say because of lack of opportunity to collect the patient's stool. (He refused to take the carbon mixture.) The potassium appears relatively increased on the fever day, but sinks next day immediately to a low figure. Calcium is scantier on the fever day than on the fever-free one. The relative increase in hydrochloric acid and the relative decrease in phosphoric acid on fever days can be readily discerned, a phenomenon that is well known to stand out more clearly if the urine for 24 hours in malaria cases is not mixed, but collected at suitably chosen intervals of time in 3 to 4 portions daily. The phenomenon of increased Cl elimination on the day of fever and diminution of phosphoric acid on the same day is a known fact and has recently been pointed out by Terray [Zeitschrift für Klinische Medicin, Vol 26] and me [Wiener Klinische Wochenschrift (Vienna Clinical Weekly), 1893] [Translator's note: Perhaps this reference should be to Wiener Medicinische Wochenschrift (Vienna Medical Weekly), 1894.]. It seems certain that

the reduction in phosphate during chills is due to retention of P_2O_5 in the body [Freund, Wiener Klinische Wochenschrift, 1893]; that the increased chloriduria is conditioned solely by the destruction of red blood corpuscles in malaria, as Terray has assumed (loc.cit.), seems to me at least improbable. The observed results thus show that in the case of febris intermittens tertiana studied all the signs of overloading of the body with acids appear on fever days, but not on fever-free days, in spite of the same nourishment on both days. In this case the feverish period of the infection is highly probably followed by over-acidification of the tissues, which however recedes on the fever-free day. This is characterized by all the urinary signs known to us, — increase in N-elimination, albeit to a slight extent; massive increase in the $N(NH_3)$, and alkali losses via the urine on the fever day, which were relatively very high.

2 and 3. Two Cases of Highly Feverish Tuberculosis of the Lungs

P. Barbara, 33-year old working girl, admitted 16 May, died 4 June. The patient, affected by a hereditary taint, suffered as a child of 10 years from what was alleged to be a bone disease, for which she underwent an operation. Ten years ago a hemoptoë occurred for the first time, recurring two years ago. Since that time the patient had suffered from "catarrh of the lungs," which she said had become especially bad since the end of January of this year. Since then night sweats and lack of appetite. Fifth childbirth five weeks ago. The child is living.

The patient is of pallid brunet complexion, slender, of flaccid musculature. Left half of thorax somewhat sunken. Clavicular fossa deeply sunken, percussion sound above it muffled. In front, in the parasternal and mamillary line, lack of resonance on the right to the fifth, on the left to the third rib. Large-bubbling râle everywhere, in all phases of respiration; at the second rib on the left, metallic breathing. The heart sounds are covered by the numerous râles. Kyphoscoliosis lumbalis sinistroconvexa; to the scapular process on each side, lack of resonance; from there down for four fingers' width on each side, moderately loud, deep tone. Râle as in front, with bronchial breathing. Numerous tuberculosis bacilli observable in the sputum. Nothing abnormal in the abdomen. No pathological substances in the urine.

Table IX shows the results of examination of the urine from 18 to 22 May of this year inclusive, 5 days, during which time the intake was accurately noted. It was impossible to study the faeces formed during this time, since the patient suffered a collapse on the last day of observation (22 May) and on 28 May determinedly refused to take the carbon emulsion a second time for the purpose of marking off the column of faeces.

The patient died on 4 June. The autopsy confirmed the clinical diagnosis.

Table IX

May	Urine	Density	N	$N(NH_3)$		KCl + NaCl	K_2O	Na_2O	CaO	U		HCl	P_2O_5	SO_3	Temperature	
			g	g	% of Tot. N				mg	g	% of Tot. N				a.m.	p.m.
18	440	1025	6.56	0.50	7.6	3.52	0.50	1.44	37	0.127	1.9	1.195	1.122	1.069	38.8	39.0
19	370	1026	5.43	0.29	5.2	3.46	0.44	1.46	64	0.113	2.1	0.968	1.11	0.858	38.0	39.0
20	365	1025	5.36	0.27	5.0	3.33	0.40	1.69	34	0.187	1.1	1.023	0.967	0.682	37.8	38.7
21	330	1020	4.36	0.24	5.5	3.26	0.39	1.39	25	0.242	1.8	0.697	1.699	1.224	37.6	39.5
22	220	1032	2.74	0.13	4.7	2.19	0.35	0.86	19	0.088	1.0	0.483	0.520	0.514	37.0	36.9
																collapse

The figures shown in Table IX are in general conspicuous for their lowness. This is true not only of the amount of urine, but also of the values for N and for the various bases and acids of the urine. If we consider especially the N values of the urine, we find a decline from 6.56 to 2.74 in the space of 5x24 hours, which must be interpreted, merely from the relatively low values in general, as an effect of undernourishment. In fact the food intake throughout the period of observation was very low, and was limited to milk, bouillon with egg, a little wine, and sometimes a bit of white bread [see note]. If we compare the total nitrogen taken in with the food with the total nitrogen eliminated in the urine, we find that with 15.7 g N taken in, 24.45 g N was eliminated, or in other words the body lost about 9 g N in that period. Whether this loss of protein came about through toxic destruction of protein as a result of existing over-acidification or could be merely the effect of hunger can be read in part from other columns of the table. Aside from the fact that the ammoniac elimination could at no time be regarded as high, but with the exception of the first day of observation on which it was perhaps slightly higher than normal, the percentage that its N represented of the total N varied always within the norm, the sum of the ingested and eliminated alkalies, at least insofar as they could be checked in the urine, also indicated no alkali losses of the body.

[Note] The N content and the KCl+NaCl content of the food taken by the patient were as follows:

18 May	2.623 g N	6.993 g KCl+NaCl
19 "	3.333 " "	7.643 " " "
20 "	3.302 " "	7.522 " " "
21 "	2.808 " "	5.805 " " "
22 "	2.683 " "	6.993 " " "
Total	15.749 g N	34.956 g KCl+NaCl

The total intake of KCl + NaCl during the 5-day observation period according to my calculation amounts to 34.95 g, while only 16.26 g KCl + NaCl left the body in the urine. It will be

seen from this, and is of importance for judging other cases, that undernourishment in man over a period of five days does not bring about an increased ammoniac or alkali content in the urine, even in spite of any high febrile rise in temperature.

As for the other figures contained in Table IX, not only the total nitrogen, but also the ammoniac, the fixed alkalies, the calcium, and the acids all show a tendency to progressive diminution, but there are also sporadic rises, which do not seem readily understandable in view of the uniformity of the intake of nourishment. Thus e.g. on 21 May there is a sudden rise in elimination of phosphoric and sulfuric acids, with a simultaneous decline in chlorides, fixed alkalies, and calcium.

3. P. Marie, 27-year-old manual worker, admitted 15 March. The patient is presumably not tainted by heredity and had previously been healthy. For three months she had had a heavy cough, with increasing loss of weight and night sweats. So far there has been no hemoptoë. Of short and slender build, much emaciated, the face reddened, visible mucous membranes pale. On the skin of the back and thorax numerous pityriasis efflorescences. Thorax poorly arched, fossae infra- and supraspinatae much sunken. Percussion reveals a damping at the right front with tympanitic accessory sound in the region of the 1st and 2nd intercostal spaces. Right rear likewise damping above, clear, full percussion sound below, left rear normal percussion conditions. In the region of the right fossae supra- and infraspinata, true amphoric respiration, with large and small bubbling râles; above the apex of the left lung at the back, rough vesicular respiration with occasional rhonchi. Heart largely covered by the left lung. Cardiac sounds pure. Abdomen shows nothing abnormal. No edema. Urine clear, contains no sugar, no protein, and little indican. Sputum mucopurulent, contains many tuberculosis bacilli.

Table X

Mar-Apr.	Amount of Urine	Density	N g	$N(NH_3)$ g	$N(NH_3)$ % of Tot. N	KCl + NaCl	K_2O	Na_2O	CaO	HCl	P_2O_5	SO_3	Temperature a.m.	Temperature p.m.
28	1200	1012	6.30	0.315	5.0	8.786	1.68	2.716	0.123	3.804	1.56	1.046	38.4	37.2
29	900	1011	5.418	0.204	3.7	5.715	1.079	2.123	0.994	2.133	1.08	0.851	37.8	38.5
30	700	1013	5.978	0.159	2.6	5.887	0.858	2.399	0.198	2.196	1.12	1.008	38.4	38.4
31	1100	1015	7.777	0.255	3.2	7.128	1.567	2.462	0.138	2.684	1.70	1.333	38.2	38.4
1	1000	1013	6.65	0.332	4.9	9.210	1.507	3.618	0.161	2.993	2.16	1.291	38.4	38.8

From study of the figures for the urine given in Table X we learn the following: The amount and the specific gravity of our patient's urine varied during the 5-day observation period within the normal limits. The N values of the urine fluctuated within a narrow range; on two days it was somewhat low [see note]. Total elimination of N through the urine amounted according to the above table to a total of 32.12 g, while the

intake of N was calculated at 37.90, so that there remains a small plus of about 5 g. According to the findings of the urine examination the patient P. thus lost no N during the time in question, at least not through the urine, even though she showed daily temperatures above normal. In harmony with this we find not only the absolute but also the relative figures for ammoniac in the urine strikingly low. A percentage of 5.5 of the total N is the highest level that the ammoniac N in the urine reached -- a figure that corresponds to the norm.

[Note] The patient's diet during the 5-day period of examination consisted of a total of 4000 ccm of milk, 2800 ccm of bouillon, 250 g of roastbeef, 436 g of white bread, and 240 g of egg. (Milk 0.419% N, 0.48% alkalies; bouillon 0.046% N, 0.583% alkalies; roastbeef 3.4% N, 1.02% alkalies; white bread 1.4% N, 1.51% alkalies; and egg 2.19% N and 0.618% KCl + NaCl.

If we go on to the columns containing the figures for the alkalies, we find not insignificant fluctuations in the total of KCl + NaCl on the individual days, which are probably due to the patient's varying need for nourishment. The total of the alkalies eliminated from the body with the urine comes to 36.726 g KCl + NaCl, while 46.13 g was ingested with the food. Considering these figures we thus come to the conclusion that our patient suffered no losses of alkalies through the urine, and this finding taken together with the earlier one that there was no excessive elimination of N and ammoniac at least rules out the assumption of the existence in our patient of any over-acidification during the five days of observation. The figures for CaO are slightly low; this might be due in part to decreased intake of food. Of the acids of the urine, the hydrochloric acid is conspicuous for its relatively too high values, while phosphoric and sulfuric acids gave low figures by comparison. In spite of this the ratio of N:P_2O_5 is slightly high in favor of the latter (100:23).

Examination of the faeces changes nothing in the above results. The faeces formed during the 5-day observation contained:

Dry matter	34.75 g
N	1.753 g
KCl + NaCl	2.56 g
CaO	2.15 g
Fat	10.79 g
HCl	0.74 g
P_2O_5	3.72 g

The N elimination thus came to a total of 33.87 g against 37.90 g of intake, thus showing a retention, though a small one. (The N content of the rather abundant sputum was not taken into account.) The elimination of alkalies is raised by the faeces from 36.72 g to 39.28 g of KCl + NaCl, while the intake came to more than 46 g.

If we take the fact that two cases of severe, progressive pulmonary tuberculosis, each of which showed high fever daily at the time of investigation, nevertheless on those days never exhibited even one of the three changes in the urine that otherwise accompany acid poisoning, then the conclusion would seem to be justified that in these two cases in spite of high fever no sign of overloading of the organism with acids existed during the period of observation. The conflict between the results of these two observations and those of the case of [febris] intermittens cannot, as I believe, be explained on the assumption that in the two cases of pulmonary tuberculosis there was a slight undernourishment and that the different behavior in these two cases was due to that (see above). Besides, Voges [cited according to von Noorden's Lehrbuch (textbook), page 168] observed, and after him von Noorden [Lehrbuch der Pathologie des Stoffwechsels (Textbook of Pathology of the Metabolism), page 168] confirmed that hunger in man raises the quantity of ammoniac relative to the total N present a little, instead of lowering it, so that even for case 1 one could assume the opposite. We must look for the cause of the lack of agreement in other factors, and particularly bear in mind the fact that fever can exist, as we have seen, without symptoms of over-acidification. In any case the connection between these two factors is not a necessary but only an occasional one.

Löwit [Vorlesungen über Allgemeine Pathologie (Lectures on General Pathology), No 1, "The Theory of Fever," Jena, 1897, pages 162 ff.] in his recently published work takes the position that the source of acid formation in fever is to be sought in the microorganisms that have invaded the body. That this assumption is possible and even that in certain cases it partly explains the occurrence of acidification has been made clear by the case of [febris] intermittens among others, where it is only by the breakdown of red blood corpuscles that acid materials are dissolved into the tissue fluids, but that is not to say that these acid products are also the cause of the thermal disturbance of the body, necessarily. But it is evident that these conditions cannot hold for all febrile states, for fever, or increased temperature, can also exist without the effect of acid poisoning, just as it has long been known that the opposite situation can occur. In the case of such patients, then, we must be concerned with the formation of one or more substances that do not lead to acid effects, and which change the thermal economy of the affected individual without particularly influencing his stock of bases and acids.

4. A Case of Measles

Bai Rifka, 8-year-old daughter of servants, from Moronetz (Bukovina). The patient was under treatment in the eye division of the institute from 10

April to 22 May for ophthalmia sympath. and on the latter day was transferred to my division because of the onset of measles. She became ill with coughing and sneezing and high fever (39.5° C), and on the morning of the 22nd already showed definite measles exanthema all over the body. Apart from this the only objective pathological indication was a trace of albumin and a little acetone in the urine. On 25 May the exanthema was still plainly visible, though the temperature had already gone down to normal. From 26 May on the exanthema had faded away, but the patient remained without appetite until 28 May, when she took the first solid food.

The patient's metabolism was studied from 24 May, the last day of fever, to 28 May inclusive, but only on the basis of the urine, as the patient refused to take the carbon emulsion.

Table XI shows the data obtained.

Table XI

May	Amount of Urine	Density	N	$N(NH_3)$		KCl + NaCl	K_2O	Na_2O	CaO	HCl	P_2O_5	SO_3	Temperature	
			g	g	% of Tot. N								a.m.	p.m.
24	1360	1004	5.18	0.56	10.8	2.42	0.30	1.03	0.208	0.127	0.646	0.565	39.1	38.5
25	1210	1008	6.52	0.67	10.2	3.49	0.24	1.64	0.077	0.135	1.421	2.520	37.0	36.4
26	1420	1004	4.12	0.37	9.0	5.67	0.07	2.92	0.062	0.256	1.136	1.775	36.9	36.2
27	1235	1010	6.39	0.79	12.3	4.46	0.55	2.54	0.061	0.146	1.883	2.747	36.0	36.2
28	875	1012	5.63	0.30	5.3	8.17	0.51	3.90	0.098	2.565	1.865	1.855	36.3	36.1

The amounts of urine stayed at approximately the same level for the first four days of observation, only to decrease on the last day, when the patient for the first time took a fairly large amount of food. The nitrogen figures for the urine on individual days differ from each other relatively little, despite the fact that N intake in the food showed not inconsiderable variations [see note]. Still we do see a slight over-elimination of N in the urine as compared to the nitrogen taken in on the first and second days and again on the fourth day. On 28 May, the last day of observation, a retention of N in the body appears very probable (intake almost 10 g, elimination in the urine about 5.6 g). A striking feature, and one that is reminiscent of findings in other infectious diseases, is the relatively high $N(NH_3)$ values, which e.g. even on the third day after recovery from fever make up 12.3% of the total nitrogen. Then if we consider the alkalies, we find an almost continuous increase in them as the observation continues, until finally on the last day of observation the highest value, over 8 g, is reached. If we compare these values with those for the alkalies supplied through the food, we find that the patient lost about 24 g through the urine and took in about 25 g of KCl + NaCl in her food. During this period of observation, then, the patient lost no alkalies through the urine, though of course that does not mean that she lost none in any way. The lack of

any examination of the faeces is to blame for this unsatisfactory situation. There is little to be said concerning the behavior of the inorganic acids of the urine. The patient's condition of hunger is clearly manifested in the chlorides, as the quantity of chlorides rises immediately by about tenfold on the day when a more varied and generous diet was supplied. Phosphoric and sulfuric acids are on the average rather decreased than increased.

[Note] The diet on 24 and 25 May consisted of 900 ccm of milk and 300 ccm of clear bouillon each day; on 26 and 27 May an eggyolk (14 g) was added to this, and on 28 May the diet consisted of 600 ccm of milk and bouillon, 50 g of rolls, 70 g chicken breast, 50 g lean ham and 110 g of egg. The nitrogen content of the food on the first two days was thus 3.90 a day, that on the next two days 4.2, and on the last day of observation 9.96 g. The alkali (KCl+NaCl) content of the food worked out as follows: first and second days 6.06 g each, third and fourth days 6.14 g each, and on the last day about 11 g of KCl+NaCl.

When we consider all these factors, especially the steadily high ammoniuria and the nevertheless low alkaluria, the question arises whether and for how long signs of over-acidification of the body are detectable in the urine. The ammoniuria extending beyond the period of fever, however, made it probable in itself in spite of the reduced elimination of alkalies that the over-acidification of the tissues lasted as long as the over-elimination of ammoniac continued.

It will be seen from this observation that our case of measles showed by changes in the urine an acidification of the tissues during the feverish period and even later. This case also shows that even with increased ammoniuria an increased elimination of the fixed alkalies in the urine can be lacking.

Concerning the cause of this increased ammoniuria in spite of the lack of any increase in alkaluria, on the basis of the literature a conjecture might be put forward that in my opinion has some probability in its favor. Gaethgens (loc.cit.) reported a dog experiment in which he fed the experimental animal with leached-out meat for some time before the administration of acid. The purpose of this procedure was to raise the ammoniac level of the animal tissue as high as possible by keeping the acid-containing salts of the meat out of the feed. Giving acid to this animal under such circumstances had little other effect than to increase the ammoniac in the urine, while an earlier dog fed with fresh, unleached meat when given acid lost both fixed alkalies and ammoniac in the urine, the former in considerable measure. It is conceivable that our measles patient for unknown reasons was either relatively poor in alkalies or rich in ammoniac, and for that reason eliminated such low amounts of alkalies and relatively high amounts of ammoniac. Since Salkowski and Munk [Virchow's Archiv, Vol 71] and recently

Beckmann [cf. E. Stadelmann, Ueber den Einfluss der Alkalien auf den Menschlichen Stoffwechsel (The Influence of Alkalies on Human Metabolism), Stuttgart, 1890] have found that the introduction of alkalies reduces the elimination of ammoniac via the urine, very probably the reverse is also true.

5. A Case of Croupous Pneumonia

S. Vincenz, 26-year-old mechanic's helper from Olmütz, admitted on the afternoon of 12 November. The patient fell ill 5 days before, with fever, chills, and stabbing pains in the right half of the chest. At the same time coughing set in, with reddish-tinged expectorate. Appetite lacking, thirst increased. Body temperature on admission 39.1° C. On the face, on the left and right upper lip, dried-up herpetic vesicles. Thorax well arched, 36 respirations a minute; the right half the thorax lags a little behind. Percussion showed at the right front a clear, full tone to the fourth, fourth and fifth ribs, from there on a bandbox tone. Normal conditions on the left. Back left percussion clear and full, right clear and full to the spina scapul., from there on muffled, at the base a bandbox tone. Breathing, back left over the apex rough and vesicular, otherwise normal; upper right also rough and vesicular, from there downwards high bronchial with occasional crepitant râle. Heart normal, pulse 116 per minute, small, weak. Spleen not palpable, liver not enlarged, nothing abnormal in the abdomen in general. Sputum rubiginous. In the urine, trace of albumin, little chloride, otherwise nothing pathological, no sediment. In the blood 23,000 leucocytes.

As to the course of the disease it may be noted that in the night of 14-15 November the critical decline of fever set in and the patient remained fever-free from that day on. On 26 November he could be dismissed as convalescent.

Table XII contains the results of the regular examination of the patient's urine. It should be mentioned, however, that

Table XII

November	Amount of Urine	Density	N g	$N(NH_3)$ g	$N(NH_3)$ % of Tot. N	KCl + NaCl	K_2O	Na_2O	CaO	P_2O_5	Cl	SO_3	Temperature a.m.	Temperature p.m.
12	900?	1024	13.48	0.70	5.1	2.90	1.231	0.503	0	2.13	0.35	1.22	—	38.8
13	1500	1023	28.45	1.47	5.1	4.57	2.886	0.869	0	2.43	0.46	1.47	37.8	38.4
14	1600	1024	32.03	1.59	4.9	5.10	1.276	1.633	0	3.12	0.44	2.50	37.9	38.3
15	1400	1024	22.54	1.37	6.0	9.71	0.334	4.867	trace	3.64	0.19	2.45	36.6	37
16	1300	1023	26.32	0.75	3.9	11.93	1.730	4.872	0.109	3.64	1.93	2.48	37.2	36.7
17	1700	1020	23.08	2.01	8.7	8.82	1.598	3.334	0.078	2.80	1.57	2.47	36.5	36.6
18	1800	1022	13.48	0.84	6.2	17.16	1.812	7.576	0.123	1.98	8.04	1.87	36.4	36.5
19	1200	1019	10.24	0.56	5.4	9.02	2.337	2.820	0.726	2.10	4.68	1.03	36.4	36.4

on 12 November the patient did not get admitted until toward noon, so that the amount of urine entered for that day as well as the absolute values found in urinalysis are not based on a full 24-hour period, and so are not directly comparable with the later figures. The quantities of urine from 13 to 19 November are relatively high, even in the period of fever (to 15 November). During that period more than 60 g of N was eliminated in the urine, while the N intake during these days (1600 ccm of milk with 0.419% N, 500 ccm of bouillon with 0.046% N, 800 ccm of lower Austrian local wine 0.017% N, and 500 ccm of wine soup 0.166% N) amounted to a total of 7.90 g N. During those days therefore quite considerable N losses from the body took place, and these lasted even into the beginning of the fever-free convalescent period up to 17 November inclusive, the patient having up to that time (excluding the first day of observation) eliminated over 132 g N via the urine and taken in a total of only 12.66 g N (milk 2.4 l, 2.6 l of wine, 1.1 l of bouillon, and 1.1 l of wine soup). On 18 and 19 November the patient was already eating meat, cooked rice, and pudding in addition to his previous diet, but the N eliminated in the urine declined. While by this reckoning the patient suffered quite considerable N losses from 13 to 17 November, that is within five days, the ammoniac presents a definite contrast to the behavior of the total N in the urine. Not only the absolute figures for ammoniac, but also its value as a percentage of the total N is hardly to be regarded as high. Even the highest value reached, 8.7% of the total N present, while in view of the existing undernourishment with food of low N content it must be characterized as pathological, would still be considered normal in a healthy person with much meat in his diet. The fact, then, that in our case of croupous pneumonia we are missing such an allegedly important sign of the existence of acidification of the tissues as the increased elimination of ammoniac, as in pneumonia, seems to occur quite frequently all round.

I myself observed simultaneously with case 5 another case of croupous pneumonia, in which similarly low NH_3 values obtained in the urine during the fever period and moderately increased quantities of ammoniac were found in the urine only during convalescence. Table XIII below contains the relevant data.

Table XIII Bauer Josef

Date	Amount of Urine	Density	Total N	N(NH_3)		Temperature	
				g	% of Total N	a.m.	p.m.
12 Nov.	600?	1022	6.84	0.24	3.5	—	38.6
13 Nov.	1600	1023	21.94	0.728	3.3	38.0	38.8
14 Nov.	1600	1024	23.74	1.315	5.5	38.4	38.7
15 Nov.	1400	1022	18.52	1.297	7.0	37.8	38.6
16 Nov.	1200	1022	17.30	1.280	7.3	37.5	37.4
17 Nov.	1600	1021	17.56	1.067	6.0	37.5	37.4
18 Nov.	1200	1018	8.4	0.798	9.5	36.6	37.3
19 Nov.	1600	1019	10.92	1.008	9.2	36.4	37.2

Similar cases are also reported, as has already been mentioned, e.g. in the works of Gumlich and Rumpf. Thus the last-mentioned author reports a case where during seven days' fever the ammoniac nitrogen never amounted to more than 2.28-7.13%. Only during the convalescence did the figures rise to 16-18% of the total nitrogen.

The fixed alkalies of the urine in case 5 amounted during the first two days (13 and 14 November), at the time when fever was still present, to a total of 9.67 g KCl + NaCl. But the intake during the same period corresponded to an alkali total of 11.91 g, so that there could be no question of any alkali losses of the body via the urine during those two fever days up to 15 November. Now if we go on to compute the alkali balance for the next three fever-free days (to 17 November inclusive), we find an elimination through the urine of 30.46 g KCl + NaCl with an intake of only 16.98 g KCl + NaCl, so that a deficit of about 13 g is left. If we subtract from this the 2.24 g KCl + NaCl that did not leave the body in the urine during the first two fever days, that leaves with respect to fixed alkalies for the period from 13 to 17 November inclusive an alkali loss for our patient of about 10 g. We thus find losses in alkalies through the urine at a time when there was no longer any fever and when there was also no increased ammoniac elimination through the urine.

In this case the difference from the norm seems to lie in the fact that the acid products of metabolism showed greater affinity to alkalies than to ammoniac, so that the former left the body in combination with them sooner than the latter. Determination of the two alkalies gave in our case, analogously with Salkowski's findings, a definite predominance of the amount of potassium over that of sodium during the first three days, which however on the third fever day (14 November) was almost balanced out and thereafter gave way to a considerable relative increase in sodium. On the last day of observation the potassium and sodium figures are almost equal. The lime of the urine was not detectable at all as long as the fever lasted, but after recovery from fever it rose again, relatively rapidly, to approximately a normal level. The elimination of the inorganic acids shows something in common in that they begin with average values or as in the case of chlorine with subnormal ones, shortly after the recovery from fever rise to higher figures, and finally fall off again somewhat. Chlorine occupies a special position in that the quantity of it eliminated is known to be always greatly reduced during the period of fever in croupous pneumonia. No basis for explaining this long known phenomenon has been gained from our observations.

From what has been said we should have grounds in our case of pneumonia (case 5) for describing it as being also accompanied by acidification of the tissues: the increased N elimination, the losses in fixed alkalies, and the slight postfebrile increase in ammoniuria tell in favor of the assumption that an over-acidification must have existed during the first six days of observation.

6. A Case of Erysipelas Faciei

M., Rosa, 21-year-old servant girl, admitted 11 January 1897. The patient according to her own account fell ill on 8 January with fever and burning pains in the face; these symptoms, she said, grew worse on 9 and 10 January and were accompanied by a considerable swelling of almost the entire face.

On the left half of the face the skin from the arcus superciliaris to the right auricula, across the cheek and the right nasolabial fold, including the bridge of the nose, much reddened, swollen, shiny. Surface slightly uneven, follicles dilated. At the periphery of the changed part of the skin, a definite wall is visible against the hair-covered scalp, the neck, and the chin. The organs of the thorax and abdomen show nothing abnormal. In the urine no albumin, no acetone, trace of indican, no reduction; temperature 39.3. During the next three days the patient constantly showed above normal temperature, though with a declining maximum. From 15 January on she was free of fever. During the period of observation no pathological components were ever found in the urine. From 18 January on the skin of the face began to scale off, and the patient was released as recovered on 22 January 1897.

As in the preceding cases, the patient's urine was carefully collected and her intake of nourishment very carefully checked as to both quality and quantity. The faeces formed by her during the six days' observation was separated into two portions by administering animal charcoal. The first dose of charcoal was given early 12 January, the second early 15 January, the day of recovery from fever, and the last early 18 January, so that it was possible to separate the faeces formed during the period of fever from that produced in the three fever-free days.

Table XIV

January	Amount of Urine	Density	N	$N(NH_3)$		KCl + NaCl	K_2O	Na_2O	CaO	MgO	SO_3	Cl	P_2O_5	Temperature	
				g	% of Tot. N				mg					a.m.	p.m.
12	920	1018	8.30	0.65	7.8	3.16	0.446	1.300	17	8	3.77	5.06	0.745	38.5	38.8
13	660	1018	7.72	0.76	9.6	3.50	0.089	1.782	2	7	1.42	4.62	0.796	38.2	39.3
14	800	1020	10.53	1.32	12.5	4.06	0.217	1.969	8	7	2.08	4.16	0.710	37.7	37.2
15	1100	1015	17.27	1.92	11.1	5.81	0.577	2.597	9	0	1.74	2.05	2.222	36.8	36.6
16	1100	1015	15.55	2.31	14.8	10.92	1.701	4.361	8	9	2.67	3.74	3.300	36.5	36.4
17	1140	1021	9.89	0.67	6.7	8.42	0.490	4.053	2	2	1.39	2.15	3.111	36.5	36.2

The results of the regular examination of the urine of this case are contained in Table XIV. If we consider especially the amounts of urine from day to day, we find somewhat fluctuating figures during the fever period, and in the fever-free period a constant though not very great polyuria. With regard to the patient's N elimination during the first three days of illness, during which there was still above-normal temperature, the following should be noted: The intake of substances containing nitrogen within these three days amounted to a total of 10.24 g N (1500 ccm of bouillon with 0.046% N; 1700 ccm of milk,

0.419% N; and 111 g of egg, 2.19% N). Comparing this figure with the total N elimination through the urine for these three days, 26.55 g, gives a nitrogen loss of the body amounting to 16.31 g for the fever period of our case. The amount of ammoniac was up a little on the first day of observation as compared to the norm, but we see that it definitely increased during the following two still feverish days, although it reached its highest level (2.31 g, or 14.8% of the total N) only on the second fever-free day (16 January). This is the phenomenon, repeatedly observed by various authors, of the lag in increased ammoniuria, which, as has been mentioned, also finds analogies in the acid experiment and in pneumonia (see above). The question of balance of the total alkalies is also of interest in our case. The intake of alkalies for the first three fever days amounted to a total of 20.41 g KCl + NaCl, while elimination through the urine amounted to only 10.72 g. It has already been brought out above how slight the rise in NH_3 content was on the first two fever days and especially on the first day. It did rise during the following days, however, and we find the same thing with regard to elimination of the fixed alkalies. We find that both the ammoniac and the fixed alkalies contained in the daily amounts of urine from 12 to 16 January were in a state of constant increase, and that not until 17 January did a sudden drop set in, in the case of ammoniac practically to a normal level, in the case of the alkalies, considering the still low intake of nourishment, to a level still high in comparison to intake. Thus if we encountered earlier a lag in increased ammoniuria until after the decline of the fever, we find here that the same circumstances may hold for the fixed alkalies, too. Now if we compute the total alkali content of the food and of the urine during the whole six days' observation, the total elimination through the urine during these six days according to Table XIV amounts to 35.87 g KCl + NaCl in all, but the intake (consisting of a total of 3500* ccm milk, 2700 ccm bouillon, 246 g egg, 40 g gruel, 300 g white bread, and 120 g of a semolina pudding) certainly exceeded that amount by a considerable figure, since the fixed alkali content of the first three foods listed here alone comes to 35.84 g, which just covers the elimination found. We thus find that in our case, in spite of the increase in the alkalies of the urine after the decline of fever, synchronous with definite increase in the ammoniac of the urine, there was no net loss of alkalies from the body through the urine. This finding, which I believe to be important to the understanding of the case under study, is also accompanied by various other phenomena which also do not seem to us to fit into the picture of acid effects. First of these is the constant predominance of elimination of sodium over that of potassium. Salkowski, who also studied a case of erysipelas faciei (loc. cit., page 223), found during the period of fever even in that

*The source has 350.0 here, but this must be a misprint.

case a plus of potassium over sodium. Another thing in our table that does not seem to correspond to the picture of acid poisoning accepted up to now is the behavior of the acids. While the chlorides during the period of fever are decidedly diminished, the phosphoric and sulfuric acids, especially the former, show quite enormously high figures, especially when we bear in mind that during those days the food intake consisted only of liquid food, poor in content. What strikes us as still more peculiar, however, is the circumstance that from the day of recovery from fever on, though on that day and the next two the intake of food was not greatly increased over that during the period of fever, phosphoric and sulfuric acids are represented by figures relatively low on the average, but the chlorides by considerably higher figures.

We thus arrive at the result that while in our case of erysipelas acid poisoning may have existed, clear indications of it are not observable in the urine until the early days of convalescence.

As mentioned above, in this case the faeces formed by the patient during the 6-day period of observation was also subjected to examination, in two parts. The first part comprised that portion of the column of faeces that was formed within the first three, still feverish, days, during which the patient had taken only liquid nourishment (1500 g bouillon, 1700 g milk, and 121 g egg). The second part of the faeces belonged to the three fever-free days of the period of observation, during which for part of the time solid food, though still only a little, was being taken into the body in addition to the liquids. During these last three days the food consisted of: 135 g egg, 1200 g soup, 1900 g milk, 40 g gruel, 300 g white bread, and 320 g cooked rice.

Table XV gives information concerning elimination in the faeces.

Table XV

Period	Dry Faeces g	N g	Fat	KCl+ NaCl	CaO	MgO	P_2O_5	Cl
3-day Fever Period	103	2.33	12.36	6.57	5.05	0.403	5.99	0.316
3-day Convalescence	19.4	1.24	3.84	5.23	2.35	0.162	1.07	0.068

A glance at this table shows important differences between the elimination of faeces in the feverish period and in the convalescent period and also permits us to discern a complete analogy with the results of the lactic acid experiment mentioned above. Only the N resorption shows a difference. First of all one is struck by the relatively greater amount of the dry matter of the faeces in the fever period as compared with the convalescent period. The intake of dry substance in the food during

this period amounted to about 363 g (bouillon 9%, milk 10.28%, egg 26% dry substance), of which 103 g, i.e. 28%, was eliminated again in the faeces. During the convalescent period the total amount of dry substance ingested amounted to nearly 600 g, and of this only 19.4 g, or about 3%, left the body in the faeces. The nitrogen, fat, fixed alkalies, lime and magnesia, and phosphoric and sulfuric acids of the faeces are high in the portion of the faeces originating in the fever period in relation to the figures for convalescence, in spite of the fact that the patient did not have solid food until the latter period. It thus seems beyond doubt that during the fever period the capacity to resorb substances of nitrogen content and also fat suffered, and that in addition inorganic bases and acids during the fever period either were retained in the faeces or secreted again from the intestinal wall. If we compare these results with those represented in Table IV, page 429 [not included in this translation], which were obtained in the lactic acid experiment, their analogy is immediately evident. In both cases the dry substance, the fat, the fixed and mineral alkalies, and the inorganic acids are increased in the acid stool, and only the N was in the other case fairly uniformly resorbed, but in our erysipelas case at the time of the acidification existing in the body was retained to a somewhat greater extent. Now if we place the results of urine analysis and faeces analysis side by side, we see that at a time when the decisive signs for acidification of the tissues were not yet present in the urine, they already existed in the faeces, — yet another piece of evidence for the view expressed above that the acid poisoning of the tissues in our case may have already existed during the period of fever, but the pathological acids left the body via the kidneys only after recovery from fever had set in.

Combining the results of my observations concerning the occurrence of symptoms of over-acidification in the metabolism in six patients with high fevers, we have in brief the following:

1. A case of <u>febris intermittens tertiana</u> showed on the fever day the pure, typical picture of over-acidification, particularly as concerns the behavior of the fixed alkalies. Albumin disintegration and increased ammoniuria also existed, though in relatively slighter degree.

2 and 3. In contrast to case 1, two cases of <u>highly feverish pulmonary tuberculosis</u> showed no signs of acid poisoning in the urine and in one case not in the faeces either, but only, especially in the first of the cases, signs of chronic undernourishment. These cases showed neither increased ammoniuria nor increased alkaluria.

4. A case of <u>measles</u> exhibited albumin disintegration and increased ammoniuria during the period of fever, but no alkali losses either during the period of fever or during convalescence.

5. A case of croupous pneumonia showed during fever great albumin disintegration but no increase in ammoniuria; the latter occurred only after the crisis. Alkali elimination was not increased during fever, either; only during convalescence did alkali losses occur.

6. A case of erysipelas faciei, which showed great albumin disintegration and somewhat increased ammoniuria during the fever, did not show any considerable increase in ammoniac in the urine until during the convalescence. No losses of alkalies took place through the urine either during the fever or in convalescence. On the other hand the stool examination gave almost a complete analogy with the acid stool in the lactic acid experiment mentioned above; this tends to show that even during the period of fever there was acid activity in the body, but that this did not become recognizable in the urine until the post-febrile period.

The conclusions that can be drawn from these observations have already been stated. It appears probable that in further clinical studies, especially of febrile cases, a still more variable picture of acid poisoning will be encountered. Not only are there cases where certainly not a single symptom of the existence of over-acidification can be found in the urine even with high fever, but also, as has been shown, not only the time of elimination of the acid products, but also their neutralization in the urine is so variable that this function is sometimes almost exclusively taken over by one factor or the other, ammoniac or alkalies, and sometimes both participate in it.

Often as over-acidification does accompany febrile processes, with varying reaction of the tissues, still fever and acid poisoning do not appear to me to be two conditions of the body which cannot exist one without the other.

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